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RESEARCH ARTICLE

Risk Factors for Pressure Ulcers in the Intensive Care Unit in the Cardiac Center

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ABSTRACT

Objectives: Pressure ulcers (PUs) can negatively affect quality of life, prolong hospital stays, and increase the costs of health care. In this study, we evaluate the risk factors for the development of PUs in patients admitted to the cardiac intensive care units (ICUs) in our hospital.

Methods: We studied 229 patients who developed PUs in the ICUs between January 1, 2020, and August 30, 2021. We obtained patient data retrospectively from physician and nurse follow-up records. We recorded patient demographic and clinical characteristics, scores from Braden Scale for Predicting Pressure Sore Risk and from the Glasgow Coma Scale (GCS), percentage of ejection fraction (EF), body mass index (BMI), operation type, indication for hospitalization, and laboratory examinations during the stages of PU formation and on which day they occurred during ICU follow-up.

Results: Patient BMI, age, and gender did not significantly differ between the groups with and without PUs ($p>0.05$). However, the percentage of EF and GCS values were significantly lower ($p<0.05$), whereas glucose, urea, creatinine, C-reactive protein (CRP) values, and mortality rate were significantly higher ($p<0.05$) in the PU group.

Conclusion: We found that the rates of PU formation and transition from Stage I to Stage II significantly increased in patients with low EF and high CRP, urea, creatinine, and glucose levels.

Keywords: Cardiac center, ejection fraction, pressure ulcers

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Introduction

Elderly patients generally have multiple comorbidities and are treated with high-risk drugs in intensive care units (ICUs). Because of long care times and treatment processes, these patients are prone to iatrogenic complications and have a high mortality risk. Approximately 22%–49% of critically ill patients experience pressure ulcers (PUs) in the ICU.[1,2]

PUs (pressure wounds, decubitus ulcers) are ulcers and necrosis that occur because of the closure of capillaries and the cessation of circulation in the skin and subcutaneous tissues due to prolonged or repetitive pressure, especially in the parts of the body with bone protrusions.[3] Risk factors for PU formation include not only immobility but also friction and shear forces, advanced age, malnutrition, anemia, prolonged length of stay in the ICU, mechanical ventilation, low scores on the Braden Scale for Predicting Pressure Sore Risk, fecal and urinary incontinence, dehydrated skin, chronic diseases, and use of vasopressors such as norepinephrine.[4,5] PUs reduce patient

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quality of life, prolong hospital stays, increase health care costs, and cause mortality and morbidity. Therefore, resolving this issue is important for both patients and the health care system.[6,7]

Hypotension, which plays a role in PU formation by impairing tissue nutrition and oxygenation, often occurs during cardiovascular surgery due to cardiopulmonary bypass and significant blood loss. Moreover, some patients cannot be repositioned in bed for hours or even days due to the support of an intra-aortic balloon pump (IABP) or other assistive devices. Therefore, PUs may occur during the ICU follow-up after cardiac surgery.[8,9] Not only patients undergoing invasive cardiac surgeries but also certain groups of patients who have diseases including heart failure and acute myocardial infarction are particularly at high risk for PUs.[10,11] Patients who develop PUs are more likely to be elderly and have high troponin I levels, low systolic blood pressure, and left ventricular ejection fraction (LVEF).[12]

In this study, we evaluated the risk factors in the development of PUs in terms of treatment and outcomes in patients who had developed PUs during ICU follow-up after cardiovascular surgery or while being followed up in the cardiology ICU due to cardiac problems.

Methods

Study Design and Patient Selection

After approval of the study by the Local Ethics Committee (23.03.2021–2021/24), we examined retrospectively 9,729 patients who had been treated in 3 different ICU departments between January 1 and December 31, 2020. The ICUs had a total of 70 beds, 32 of which were in the postoperative cardiovascular surgery (CVS) follow-up unit, 26 of which were in the cardiology ICU, and 12 of which were in the CVS isolation unit. We found PUs in 229 of the 9,729 patients; 6,134 patients were treated in the cardiology care unit, and 3,595 were followed up in the CVS ICU postoperatively.

PUs usually develop after a hospitalization period of >3 days.[13] We excluded patients with a hospital stay of <72 h and those <18 years of age from the study. This study was conducted according to the Declaration of Helsinki.

As a routine practice, our hospital uses air beds in the ICUs; the wound care nurse makes daily visits and records patient notes. To prevent PUs, patient bed position is routinely changed every 2 h, and protective silicone pads are used for heels. Inotrope and vasoactive inotrope scores as stated in the sepsis and cardiogenic shock guidelines are followed. The hospital uses the Braden Scale to assess PU risk.

Patient PU stages are evaluated according to the hospital's Pressure Ulcer Risk Prevention/Treatment Form (Braden Scale) and recorded in patient files. We examined the demographic data of patients who developed PUs in the ICU, including the number of days in the ICU, the first day on which the PU developed, the approach to care, and any wound infections. We recorded patient age, gender, and diagnosis; name of the ICU and duration of patient follow-up; PU formation risk; PU stage and anatomical localization; duration of hospital stay before PU formation; levels of hemoglobin, glucose, albumin, urea, and creatinine; and platelet counts on the day of formation. We also noted the percentage of ejection fraction (EF), GCS scores, the presence of diabetes mellitus, and body mass index (BMI).

Definitions

Number of inpatients. The number of patients treated in the ICU within a certain period (day, month, 3 months, or 1 year) were included.

Pressure Ulcer Risk Scale: Questions were based on the hospital's patient diagnosis form, the Braden Scale, the European Pressure Ulcer Advisory Panel's staging form, and the diagnosis form for patient general disease status. The questionnaire included patient age, gender, and sociodemographic characteristics; medical diagnosis during ICU admission and length of stay; use of sedation; nutritional status; vasopressor intake; and frequency of positioning, care, and treatment interventions. This form was completed daily in line with the information obtained from the follow-up and nursing care form used in our unit.

Braden Risk Assessment Scale: Bergstrom et al.[14] developed this scale to consider patient PU risk factors. Although the Braden Scale was designed for the general patient population, it is widely used in ICUs,[15] mainly because its application requires little time to complete and so provides a quick assessment, an advantage in this environment. However, the scale has some disadvantages: its scope is limited to patients in ICUs; raters can make a subjective interpretation, leading to differences among raters; and most patients are considered to be at risk for AIDS.[16] The Braden Scale has a high predictive value in the evaluation of PUs, with scores ranging from 6 to 23, and 6 main headings, including mobility, activity, sensory perception, nutrition, moisture, and friction and shear, are evaluated in scoring.[14,17]

Pressure ulcers: PUs are classified into four stages according to the clinical manifestations on the basis of recommendations from the National Pressure Ulcer Advisory Panel:[18]

- Stage I: Skin is intact, but erythema does not disappear when the existing pressure is removed.

Table 1. Patient minimum, maximum, and median values for the study parameters

	Min-max	Median	Avg.±SD	n	%
Age	29.0–98.0	68.0	68.0±12.1		
Gender					
Female				89	38.9
Male				140	61.1
Stage I (day)	2.0–130.0	13.0	17.3±18.5		
Stage II (day)	3.0–140.0	18.0	23.9±23.3		
Difference	1.0–50.0	3.0	5.0±8.2		
BMI	16.5–40.4	27.7	28.4±4.9		
GCS	3.0–15.0	9.0	9.7±4.7		
HGB	6.6–20.0	9.0	9.5±2.0		
PLT	6.8–671.0	176.0	178.6±130.5		
Albumin	13–65	27.05	27.54±7.3		
Glucose	70.0–812.0	145.0	177.6±93.0		
Urea	5.0–150.0	38.0	45.8±26.8		
Creatinine	0.2–9.7	1.5	1.8±1.4		
CRP	1.0–396.5	89.7	107.4±84.9		
Braden	7.0–19.0	12.0	12.1±2.7		
EF%	20.0–70.0	50.0	46.3±13.3		
Increase in ulcer					
No				83	36.2
Yes				146	63.8
Stage					
I				69	30.1
II				155	67.6
Unknown				5	
Stage-I					
≤11 day				108	47.1
>11 day				121	52.9
Exitus					
(–)				122	53.2
(+)				107	46.8

Min: Minimum; Max: Maximum; Avg: Average; SD: Standart deviation; BMI: Body mass index; GCS: Glasgow coma scale; HGB: Hemoglobin; PLT: Platelets; CRP: C-Reactive Protein; EF: Left ventricle ejection fraction.

- Stage II: Partial skin loss and bulla formation are observed.
- Stage III: Subcutaneous tissue and muscle layers are involved.
- Stage IV: Bone and joint involvement are detected.

Statistical Methods and Analyses

We used the G*Power (v3.1.9) program to determine sample size. Power analysis can be performed between measurements and at different times. Even changes with a small effect size (dz=0.200) will occur. We carried out our analysis to determine the minimum number of cases required to show statistical significance. Accordingly, 80% power at the level of α=0.05 indicated we should have at least 199 cases in the study.

For the descriptive statistics, we used mean, median, lowest and highest values, standard deviation, frequency, and ratio values. We measured the distribution of variables with the Kolmogorov–Smirnov test. We used the Mann–Whitney U test to analyze the quantitative independent data and the chi-square test to analyze the qualitative independent data. We used the SPSS 28.0 program during the analysis.

Results

Table 1 shows the minimum, maximum, and median values for the parameters we studied. Table 2 shows the localizations of PU development in the patient groups.

Age and gender distribution; BMI; GCS score; platelet count; albumin, glucose, urea, creatinine, and C-reactive protein (CRP) levels; Braden Scale scores; percentage of EF; stage distribution; increased wound rate; and incidence rate of exitus did not differ significantly in the

Table 2. Pressure ulcer localization

Body part	n	%
Sacrum	161	70.3
R Gluteal	14	6.1
L Gluteal	11	4.8
R Trochanter	10	4.3
Coccyx	8	0.4
R Scapula	8	3.4
L Trochanter	4	1.7
L Scapula	3	1.3
L Heel	2	0.8
Nape	1	0.4
R Thorax	1	0.4
R Elbow	1	0.4
R Heel	1	0.4
Scapula	1	0.4
Scrotum	1	0.4
Back	1	0.4
L Ear	1	0.4

R: Right; L: Left.

groups with Stage I PU formation of <11 days (mean days of patients with and without increased scar, according to the study) and of >11 days (p>0.05). Hemoglobin levels in the group with Stage I PU formation of >11 days were significantly lower than were those in the group with Stage I PU formation of <11 days (p<0.05) (Table 3). We performed the chi-square test for EF of <35% and >35% and for PU formation of <11 and >11 days but found no significant relationship (p=0.418) (Table 3).

We evaluated the patients with Stage I PUs in two groups. In Group 1, wound size increased and stage advanced, and in Group 2, wound size did not increase and remained at Stage I. Patient age, gender, BMI, hemoglobin and albumin levels, platelet count, and Braden Scale scores did not differ significantly between the two groups with and without wound enlargement (p>0.05). In Group 1, GCS scores (Fig. 1) and EF percentages were lower, but glucose (Fig. 2), urea (Fig. 3), creatinine (Fig. 4), and CRP (Fig. 5) values were higher than were those in Group 2 (p<0.05).

Table 3. Study parameters for patients with pressure ulcers forming at <11 and >11 days (mean days with and without increased scar)

	Stage-I; PU ≤11 days				Stage-I; PU >11 days				p	
	Avg.±SD	Median	n	%	Avg.±SD	Median	n	%		
Age	68.3±13.1	68.5			67.8±11.1	68.0			0.870	m
Gender										
Female			31	37.3			38	40.4	0.675	X^2
Male			52	62.7			56	59.6		
BMI	28.8±4.9	28.3			28.0±4.9	27.7			0.357	m
GCS	9.6±4.9	8.0			9.9±4.5	10.0			0.625	m
Hb	9.8±1.9	9.3			9.2±2.1	8.9			0.004	m
PLT	199.4±132.3	182.0			160.2±126.7	150.5			0.051	m
Albumin	27.83±6.18	27.46			27.3±8.1	25.6			0.126	m
Glucose	176.2±79.0	159.0			178.8±104.1	139.0			0.745	m
Urea	44.0±26.5	35.0			47.3±27.1	41.0			0.460	m
Creatinin	1.8±1.4	1.5			1.8±1.4	1.4			0.858	m
CRP	111.9±88.1	97.1			103.4±82.2	84.0			0.672	m
Braden risk score	12.2±2.9	12.0			12.0±2.4	12.0			0.961	m
EF%	45.5±14.2	50.0			46.9±12.5	50.0			0.681	m
Stage										
I			36	37.9			34	25.4	0.067	X^2
II			59	62.1			100	74.6		
Increase in ulcer										
No			44	41.9			40	32.3	0.211	X^2
Yes			61	58.1			84	67.7		
Exitus										
(-)			63	57.8			59	49.2	0.179	X^2
(+)			46	42.2			61	50.8		
EF %										
≤35			31	29.9			30	24	0.418	X^2
>35			73	70.1			95	76		

PU: Pressure ulcer; Avg: Average; SD: Standart deviation; BMI: Body mass index; GCS: Glasgow coma scale; Hb: Hemoglobin; PLT: Platelets; CRP: C-Reactive Protein; EF: Left ventricle ejection fraction.

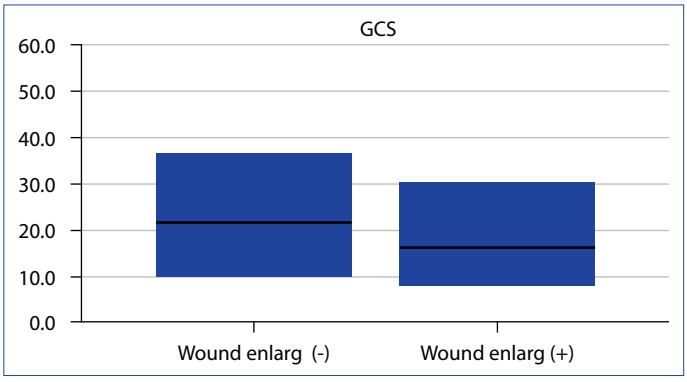

Figure 1. Patient Glasgow Coma Scale scores--Wound Enlargement Association.

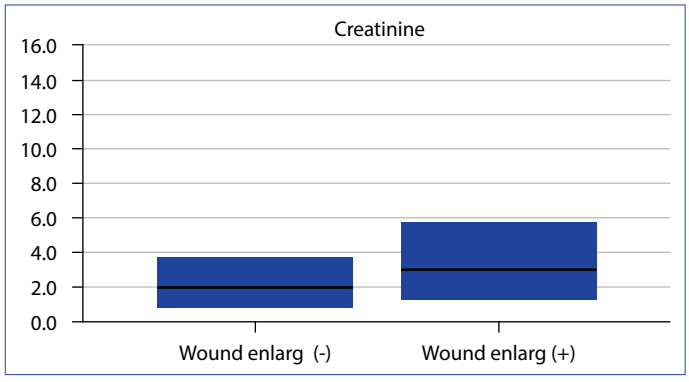

Figure 4. Patient creatinine values--Wound Enlargement Association.

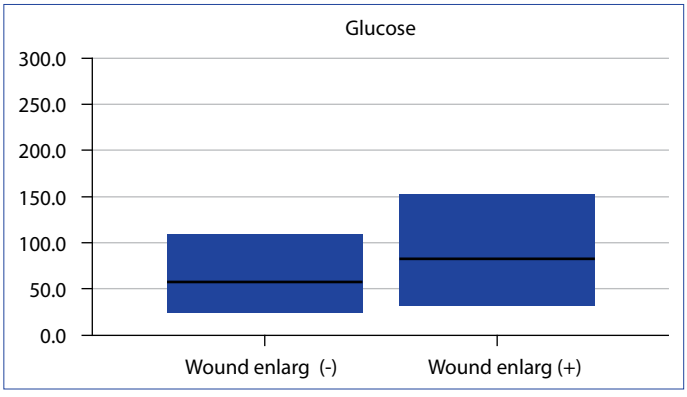

Figure 2. Patient glucose values--Wound Enlargement Association.

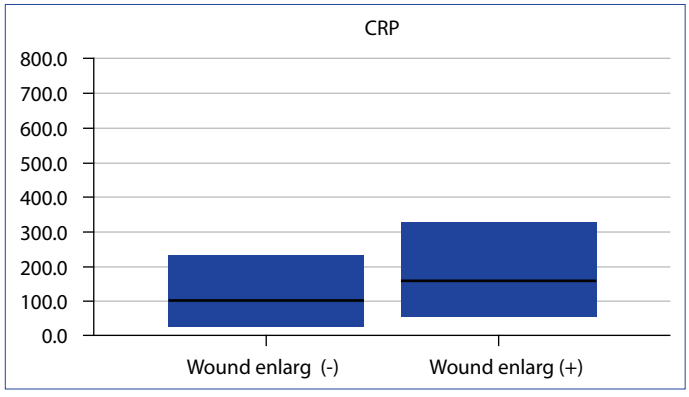

Figure 5. Patient C-reactive protein values--Wound Enlargement Association.

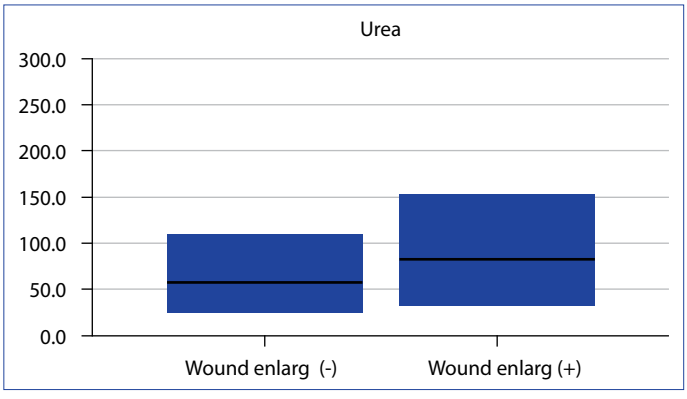

Figure 3. Patient urea values--Wound Enlargement Association.

We used the chi-square test to investigate the relationship between the groups with and without wound enlargement in terms of percentage of EF. When EF was <35%, wound enlargement and increase in ulcer stage was significant ($p<0.05$) (Table 4, Fig. 6). Group 1 with wound enlargement was significantly higher than Group 2 without wound enlargement in terms of stage ($p<0.05$) (Fig. 7). The rate of duration of <11 or >11 days did not significantly differ between these groups ($p>0.05$). The mortality rate for Group 1 was significantly higher than was that for Group 2 ($p<0.05$) (Fig. 8).

Discussion

PUs commonly develop in immobile patients in ICUs or in those who are bedridden and can prolong the hospitalization period and cause financial and labor time losses.[19] Each year, more than 1 million people develop PUs in the United States, and this risk is particularly high in patients with cardiovascular disease.[20,21]

We defined chronic heart failure as an important risk factor for the development of PUs after cardiac surgery. In addition, the complex procedures performed during surgery, the hypotension that develops during surgery and is sustained, the long periods of paralysis and heavy sedation, hypothermia, and use of vasopressor drugs also can affect PU development.[22] In our study, we found that PUs were more common in patients with an EF of <35%, either followed up in the cardiology ICU or postoperatively in the CVS ICU (Table 4). Therefore, LVEF should be considered when evaluating PU risk factors in ICUs. IABP was not used in any of our patients.

Malnutrition can impair blood flow, immune response, and peripheral oxygenation, leading to impaired wound healing.[23] Sugino et al.[24] have argued that serum albu-

Table 4. Selected study parameters for patients with and without wound enlargement

	Wound enlargement (-) Remaining in stage-I / Group 2				Wound enlargement (+) Increasing stage/ Group 1				p	
	Avg.±SD	Median	n	%	Avg.±SD	Median	n	%		
Age	67.1±13.4	68.0			68.5±11.3	69.0			0.622	m
Gender										
Female			28	43.8			41	36.3	0.328	X^2
Male			36	56.3			72	63.7		
BMI	27.7±5.3	27.5			28.8±4.7	27.8			0.137	m
GCS	10.8±4.7	11.5			9.1±4.6	8.0			0.014	m
Hb	9.2±1.4	8.9			9.7±2.3	9.1			0.477	m
PLT	188.3±147.4	171.5			173.1±120.3	176.0			0.654	m
Albumin	27.4±6.6	27.3			27.7±7.7	26.9			0.646	m
Glucose	159.3±76.1	126.0			188.0±100.1	148.0			0.013	m
Urea	38.8±27.1	31.0			49.8±25.9	49.0			0.001	m
Creatinine	1.7±1.7	1.1			1.9±1.2	1.6			0.001	m
CRP	89.8±80.6	69.7			117.3±86.0	99.8			0.020	m
Braden R. S	12.6±2.8	13.0			11.8±2.6	11.0			0.076	m
EF%	54.6±10.3	59.0			41.8±12.5	40.0			0.000	m
Stage										
I			60	69			13	9.2	0.000	X^2
II			27	31			129	90.8		
Stage I (days)	14.3±14.0	11.0			18.9±20.5	14.0			0.088	m
Ulcer										
Stage-I										
≤11 days			44	52.3			63	43.4	0.211	X^2
>11 days			40	47.7			82	56.6		
Exitus										
(-)			59	69.4			64	44.4	0.002	X^2
(+)			26	30.6			80	55.6		
EF%										
≤35			7	8.5			55	37.4	<0.000	X^2
>35			75	91.5			92	62.6		

Avg: Average; SD: Standart deviation; BMI: Body mass index; GCS: Glasgow coma scale; Hb: Hemoglobin; PLT: Platelets; CRP: C-Reactive Protein; EF: Left ventricle ejection fraction.

min level is not a good predictor for PU formation but instead is a good prognostic indicator for wound healing. We observed in our study that the average albumin level of patients with PUs was 26.9 mg/dl and 27.3 mg/dl in patients without an increase in the PU, which was not statistically significant (Table 4). The minimum value for albumin was 13 mg/dl, the maximum was 65 mg/dl, and the median was 27.05 mg/dl (Table 1).

Researchers have reported that PUs are most commonly observed in the sacral region.[25,26] Similarly, in our study, we determined that the PUs developed mostly in the sacrum. We observed PUs in the sacral (161, 71%), right gluteal (14, 7%), left gluteal (11, 5%), right trochanteric (10, 4.5%), coccygeal (8, 3.4%), and right scapular (8, 3.4%) regions (Table 2). Therefore, it is critical to identify and closely monitor patients, especially those most at risk. Although it is necessary to use PU staging for identification and to perform appropriate care intervention for wound type using the correct materials, this is not easy because of the differentiation in PU development. In our study, we observed that the wounds in the sacral region were mostly at Stage II, despite the precautions taken and regular follow-up by the wound care nurse. However, our patient population was elderly and had multiple comorbidities.

Ranzani et al.[27] also determined that high glucose and low hemoglobin levels were associated with PUs. In our study, the hemoglobin values in the group with Stage I PUs of >11 days was found to be significantly lower than that in the group with Stage I PUs of ≤11 days ($p<0.05$) (Table 3).

Many studies have shown that PUs are related closely to low GCS scores and anemia, but no significant rela-

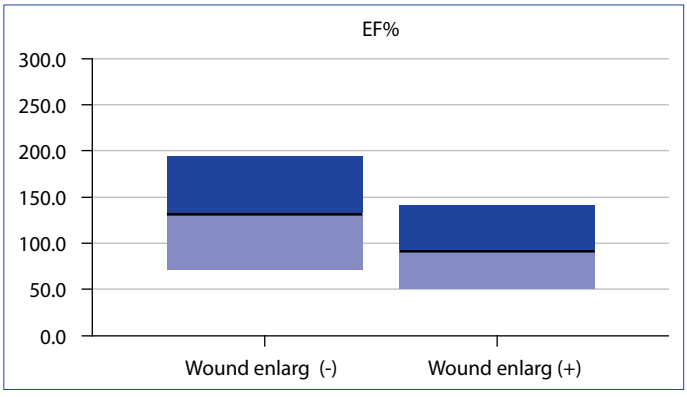

Figure 6. Patient ejection fraction percentages--Wound Enlargement Association.

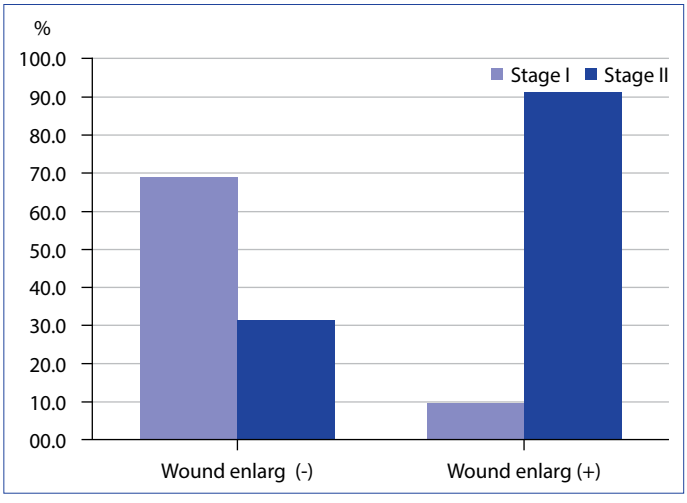

Figure 7. Patient wound stages--Wound Enlargement Association.

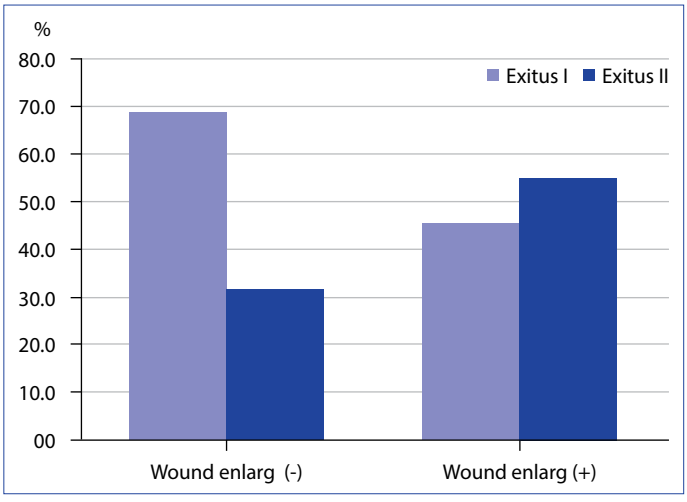

Figure 8. Patient exitus--Wound Enlargement Association.

tionship has been established with age, gender, surgical procedure, tracheostomy, prolonged fever, and albumin changes.[28] In our study, we determined that the increase in PU stage was faster in the patients with low GCS scores and hemoglobin values.

The risk of bacteremia is higher in patients who develop PUs.[29] In our study, we found that the CRP value, which is an acute phase reactant, was significantly higher in the group with wound enlargement than in the group with no wound enlargement ($p<0.05$).

Although some studies in the literature have shown that gender has no effect on PU development,[30] others have advocated that almost twice as many PUs occur in women.[31] In our study, we found no significant difference in terms of gender (Table 3).

In hospitalized patients with PUs, additional comorbidities such as acute renal failure, chronic renal failure, and pneumonia may be present.[32] Frankel et al.[33] have defined diabetes and renal failure as independent risk factors for developing PUs. In our study, urea and creatinine values were significantly higher in the group with wound enlargement ($p<0.05$) (Table 4).

In addition to cardiovascular interventional strategies, a care plan based on individual mobility should be developed in elderly patients with reduced cardiac function. Larger multicenter studies are needed to better understand how EF can help identify patients at high risk for PUs.

In conclusion, PU development must be prevented due to the burden on both patients and the health care system. In our study, we found that the rate of formation and the transition of PUs from Stage I to Stage II significantly increased in patients with low EF percentages and with high CRP, urea, creatinine, and glucose levels. PUs are common but preventable in hospitals and ICUs, and according to our study, they can be prevented with accurate estimation. However, the first step for successful prevention is to identify the main factors that predispose patients to development. ICU physicians, nurses, and other health workers must be aware of the risk factors that play a role in development and must make appropriate interventions in time for prevention.

Disclosures

Ethics Committee Approval: The study was approved by The Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital Clinical Research Ethics Committee (Date: 23/03/2021, No: 2021/24).

Informed Consent: Written informed consent was obtained from all patients.

Peer-review: Externally peer-reviewed.

Conflict of Interest: None declared.

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Authorship Contributions: Concept – B.B., G.T.G.; Design – B.B., G.T.G., Z.Ö.U.; Supervision – B.B., G.T.G., Z.Ö.U., M.C.K., A.O.B.; Fundings – B.B., M.C.K., A.O.B.; Materials – B.B., G.T.G.; Data col-

lection &/or processing – B.B., M.C.K., A.O.B.; Analysis and/or interpretation – B.B., M.C.K.; Literature search – B.B., G.T.G., Z.Ö.U., M.C.K., A.O.B.; Writing – B.B., A.O.B.; Critical review – B.B., G.T.G., Z.Ö.U., M.C.K., A.O.B.

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